VOLUME 26 JULY 1940 NUMBER 7

PROCEEDINGS
OF THE
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Publication Office: Mack Printing Company, Easton, Pa.
Editorial Office: Harvard School of Public Health, Boston 15
Home Office of the Academy: Washington, D. C.

Entered as second-class matter June 21, 1926, at the Post Office at Easton, Pennsylvania, under the Act of August 24, 1912, authorizing the inclusion of a special rate of postage provided for in section 1103, act of October 3, 1917, as amended June 15, 1918.

Published Monthly

Price per annum, $5.00

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PROCEEDINGS
OF THE
NATIONAL ACADEMY OF SCIENCES

Volume 36 July 15, 1950 Number 7

A NEW TYPE OF SELF-STERILITY IN PLANTS

By Ernest B. Babcock and Morris B. Hughes

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Read before the Academy, April 24, 1950

There are two well-known genetical systems controlling self-sterility in plants that is caused by incompatibility between a plant's own pollen and its pistil. One of these systems is associated with the phenomenon of heterostyly as reported in *Lythrum*, *Linum*, *Primula*, etc., by Darwin[1] and others. Lewis,[2] in his recent review, shows that the system found in heterostyled plants is characterized as follows: (1) One or two genes with two alleles; (2) coordinated gene action in pistil and pollen; (3) diploid (sporophytic) pollen control; (4) dominance.

In contrast to heterostyled plants, most homostyled plants, for example, *Nicotiana*, have the following system: (1) One gene with multiple alleles; (2) independent gene action in the pistil; (3) haploid (gametophytic) pollen control; (4) no dominance.

One previously reported exception among homostyled plants is found in *Capsella grandiflora* which possesses the system of heterostyled plants.

The present paper reports the discovery in two members of the Compositae of an entirely new system which is a combination of the first two features of homostyled plants and the last two features of heterostyled plants, as follows: (1) One gene with multiple alleles; (2) independent gene action in the pistil; (3) diploid (sporophytic) pollen control; (4) dominance in the anthers.

That some such new system exists in *Crepis* was first suspected by Babcock and Cave[3] in 1938. Their preliminary studies indicated that in *Crepis foetida* the genetical basis of self-sterility probably did not conform to either of the previously known schemes. The following year the junior author of the present paper undertook research on self-sterility in one subspecies of *Crepis foetida*. Although his doctoral thesis[4] was accepted in 1943, his report[5] is not yet published. It will appear, as a joint publication with the senior author of the present paper, in the September number of

Genetics. Meanwhile, Gerstel[6] has discovered that the *Crepis* system also exists in the well-known rubber plant, Guayule (*Parthenium argentatum*). His report of this discovery will appear in the July number of *Genetics*.

The genetical system controlling self-sterility in *Crepis* and *Parthenium* is unique in that it is a combination of certain of the features characterizing each of the two previously known systems. The theoretical possibility of the existence of such a system for the control of self-sterility was recognized by Lewis[2] in 1944, but its existence was then considered by him as hardly to be imagined.

The only serious difficulty concerns the second and fourth features of the new system: independent gene action in the pistil and dominance of certain alleles in the anthers. It was noted by Lewis,[2] however, that the immunity type of reaction involved in the physiological concept of the oppositional relation between alleles for self-sterility provides for the variation necessary to meet the requirements of a large multiple allele series together with the specificity necessary to provide for independent gene action in the pistil. Since the specificity of proteins is practically unlimited, is it not conceivable that in the Compositae, the most highly evolved of all flowering plants, protein relations have developed which provide just the types of specificity required for the independent action of alleles in the pistil and for diploid pollen control combined with dominance of certain alleles in the anthers?

In *Crepis* a plant's own pollen fails to germinate on the stigmatic surface. This reaction is obviously between two cytoplasms, both of which are under the influence of the same gene, or (as Lewis[7] recently postulated) of two self-reproducing parts of a gene, between which no crossing over occurs. Lewis[7] also reports that radiation experiments show that the S-gene behaves toward X-rays as though it were two independent units, one determining pollen reaction, the other, style reaction.

This concept that the S-gene may behave as a "dual" entity deserves further investigation.

[1] Darwin, C., *The Different Forms of Flowers on Plants of the Same Species*, D. Appleton & Co., New York (1896).
[2] Lewis, D., *Nature*, **153**, 575–582 (1944).
[3] Babcock, E. B., and Cave, M. S., *Z. Ind. Abs. Vererb.*, **75**, 124–160 (1938).
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[7] Lewis, D., *Biol. Rev.*, **24**, 472–496 (1949).

"REPEATS" AND THE MODERN THEORY OF THE GENE

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Communicated May 5, 1950

Bridges introduced the term "repeats" for those sections of the salivary gland chromosomes of Drosophila which seem to be completely identical with other sections, the minimum extent of a repeat being a single band. He assumed that a section had once been actually reduplicated and had been inserted into the normal structure of the chromosome as a repeat, tandem or otherwise. He went one step further by suggesting that such repeats might account for the origin of new genes, if the repeated "gene" would change its function in the new position. Because the origin of new genes is completely mysterious, and because phylogeny without some such process is hardly conceivable within the framework of the gene theory, many geneticists have been found willing to accept this suggestion. This is rather surprising because the assumption is irreconcilable with the basic facts and tenets of classical genetics. A gene is supposed to reproduce its kind except when it mutates. The mutant gene, i.e., a member of a pair of alleles, has an action different from that of the original gene, and the same is true for any number of mutational changes, i.e., multiple alleles, but always affecting the same kind of process which, by way of extrapolation, is assumed to be controlled also by the original gene. Genes have never been known to mutate at different occasions into different directions, only into different grades of one effect. There is only one case known (the alleles spineless and aristapedia in *Drosophila melanogaster*) in which this relation does not seem to hold. To assume that a "repeated" gene can develop into a completely new type of gene, amounts, against the background of the classical conception of the gene and the facts of genetics, to mysticism. If it is pointed out that the new position of the gene makes a new effect possible—a vague allusion to the position effect—this is again an assumption which contradicts all known facts. Position effect produces the phenotype of a mutant of an adjacent locus (where known) as dominant or recessive effect, or, in special cases, as a mosaic effect. No fact is known which would justify the assumption that a change of position could make a known locus act otherwise than by producing its typical mutant effect, which includes the effects of multiple alleles.

Recently a number of facts have come to light which have been accounted for by the assumption of "repeats." The general type of these facts is this: Two or more mutants are found which behave like multiple alleles. Both produce, if homozygous (recessives), a definite effect, similar

but slightly different for each. In a compound the same effect is produced, which points to the presence of three multiple alleles. But careful experimentation reveals crossing over between the mutants, all other interpretations being excluded. Thus they behave as different "genes," though acting as alleles (pseudo-alleles, Lewis), and the conclusion is reached that they originated as repeats of one pre-existing gene. A number of such cases have now been studied (Lewis, Laughnan, Green and Green, Raffel and Muller, Komai) and others are suggested (Dunn and Caspari). An analysis of the facts, especially those unearthed in the remarkable work of Lewis and the Greens is apt to show the difficulties which the classical theory of the gene has to face and the superiority of a more modern concept.

Because Green and Green's work has thus far gone furthest inasmuch as a set of three "repeated" loci was found, we may use it as the basis of discussion. The decisive points are these: Among many lozenge alleles, all of which affect the quantity of the eye pigment, the eye structure, and the absence of the female spermathecas, and all of which behave as a typical series of multiple alleles, three could be shown thus far to exhibit a small amount of crossing over (resulting in one normal chromosome and one with more than one lozenge locus.) Thus they behave like individual loci, in close proximity, but permitting cross-over breaks between them. Nevertheless they act as alleles. This is best realized if different combinations are compared with at least one allele in both chromosomes with those in which one, two, or three alleles are present only in one chromosome. In a standard case of Mendelian inheritance individuals $\frac{a\ +\ c}{+\ b\ +}$, $\frac{a\ b\ +}{+\ +\ c}$ or $\frac{a\ b\ c}{+\ +\ +}$, etc., should all be normal. But here only the last one is normal, the others show the compound lozenge effect, i.e., *a*, *b* and *c* behave as pseudo-alleles. The assumed "repeats" thus break the elementary rules of genetics and Green and Green know of no way out but to assume that in the first two cases the *b* or *c*, if separated from its mates, produces a position effect, thus giving the idea of position effect a quite new and doubtful definition, which was possibly suggested by a superficial resemblance to the original Bar position effect.

In my opinion these facts fit simply and without any new assumption into the modern picture of the basic features of the chromosome at the "genic" level. A group of facts are known (see especially Demerec 43 and Goldschmidt 44, full review and discussion in Goldschmidt 49) which show that the real genetical units of the chromosome are sections of different size, containing a number of bands in the salivary chromosomes, the maximum of which is not yet known. These units are characterized by the fact that whatever happens within this section produces a mutant effect of the same kind and that all these effects behave as multiple alleles. If we take, e.g.,

the "yellow" section invisible changes of a smaller order than one band, so-called point mutations, produce the mutant yellow. Translocation, inversion or deficiency breaks within this section act like mutants (position effect), also producing yellow, and all the point mutants and position effects behave as a series of multiple alleles. One may conclude that the invisible changes, the point mutants, are therefore also rearrangements, but within a single band. Whether this conclusion is drawn or not, it becomes clear that the whole section acts in some respects as a unit. Whatever happens within it, produces the same effect, or one very similar. If we should try to account for these facts by means of the classical theory of the gene, the whole section should be the gene because all changes within it are allelomorphic. The individual bands and their invisible mutations would become subgenes. But the position effects would also be subgenes. In addition, crossing over within the section seems possible. This is very unsatisfactory and the conclusion is obvious that at this level the classical theory of the gene does not work (see my former papers loc. cit.).

If we return now to the work on "repeats" it is seen to fit very easily into the group of facts and the concept just reported. At a former occasion (loc. cit.) I mentioned a disagreement between Muller and Demerec in regard to which band in the salivary chromosome should be regarded as the yellow locus. (There are similar discrepancies for other loci; see Bridges and Brehme, 1944.) The facts just reviewed led to the conclusion that there is no reason why both these authors should not be right. An invisible mutant change (point mutation) in any band of the yellow section would produce yellow. There can be at least as many point mutants of the same kind (and allelic) within any such section as there are bands. The application of this conclusion to the lozenge case is obvious: The lozenge effect is localized in a section which contains at least three bands which can mutate as so-called point-mutations. (Additional position effect alleles are bound to be discovered.) All must have a lozenge effect, all must be allelic just as it is proved for the yellow and scute sections, etc. (see Demerec and Goldschmidt, loc. cit.). No "repeats" are needed and no position effects. All facts fall in line simply if we forget about the classical theory of the gene and look at the facts dispassionately. Actually the work on the so-called repeats is a new proof of the correctness of that part of the newer ideas concerning the basic elements of genetics, which has been discussed here.

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ABSENCE OF MUTAGENIC ACTION OF X-RAYED CYTOPLASM IN HABROBRACON

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Communicated by C. W. Metz, May 15, 1950

The literature concerned with x-ray effects on cells includes few records of attempts to separate injury induced in chromosomes from that induced in cytoplasm. Vintemberger[1] working with frog eggs concluded that it is the nucleus of the cell which is sensitive to x-rays. "L'irradiation de la région nucléaire a donc les mêmes effets que l'irradiation de la cellule entière." Dose used was 115 r. Zirkle[2] found that injury to fern spores by α-particles can be induced by extra-nuclear irradiation alone if dose is sufficiently large but that it is much greater when the nucleus is treated Astaurov[3] obtained androgenetic males from x-rayed Bombyx eggs fertilized by untreated sperm. These males (from untreated chromosomes in treated cytoplasm) were normal and their production continued after doses completely lethal to the expected types of progeny, biparental males and females. Henshaw[4] found a direct correlation between the presence of a nucleus at time of irradiation and the manifestation of an effect, delay in cleavage. He worked with nucleated and non-nucleated fragments of Arbacia eggs. Petrova[5] compared results of exposure to α-particles of entire cells of the alga Zygnema with those obtained by the treatment of the cytoplasm alone. She found that the mean lethal dose of the former ("Kerntod") was to that of the latter ("Plasmatod") as 1 to 700. Types of response differed under the two conditions of treatment. Transmissible changes were induced only when the entire cell had been irradiated.

What appears to be a striking exception to the conclusions of these inves-

tigators is the behavior of Amphibian eggs x-rayed in the germinal vesicle stage. Duryee[6-8] found that high doses of x-rays caused fragmentation of egg chromosomes, loss of lateral loops and separation of synaptic pairs as well as injury to the nucleolus when the nucleus was irradiated in the cell. Nuclei did not react markedly when irradiated after removal from the cell. Intermediate degrees of nuclear damage were produced by exposure of non-irradiated nuclei to x-rayed cytoplasm or to alkalinized Ringer's solution. These injuries appeared immediately following "as little as 30,000 r." Some changes could be detected with "as little as 10,000 r." Duryee concludes that "These data are consistent with a hypothesis that x-ray damage to nuclear components is not primarily a direct effect but an indirect one, probably caused by chemical changes in the cytoplasm."[8]

A consistent factor in the work of these authors is the high dose required for the induction of effect through cytoplasmic change. Duryee's statement that injuries can be induced by doses as low as 10,000 r stands in marked contrast to the reports of the cytogeneticists, most of whom work with relatively low doses. In fact, detailed analysis of chromosome changes induced by 50 r has been made several times.

Exceptionally favorable material for a further study of this problem is provided by Habrobracon. Normally, unfertilized eggs of this wasp develop into haploid males which are, of course, gynogenetic, while fertilized eggs develop into diploid biparental females. By suitable treatment with x-rays the chromosomes of an unfertilized egg can be made non-functional so that after fertilization the treated egg will develop as a haploid androgenetic male with paternal chromosomes only. If untreated sperm are used the possible mutagenic effects of the x-rayed cytoplasm on the untreated chromosomes can be tested. As indicated below, the experiments fail to show any evidence of mutagenic action on the part of the irradiated cytoplasm. Cytoplasm can be so seriously damaged by very high doses of x-rays as to prevent development, even of androgenetic males. The few androgenetic males surviving as this degree of injury is approached afford no evidence for mutagenic effect. This injury to Habrobracon cytoplasm is, in the opinion of the author, of the same nature as that induced by high doses administered to the cytoplasm and described in the works of the authors cited above.

The experiments on Habrobracon were conducted as follows: unmated homozygous wild type females were x-rayed and then mated to untreated males with one or more traits recessive to wild type. Dose ranged from 1350 r to 54,075 r. Three types of progeny were produced, the expected wild type biparental females and gynogenetic males and the exceptional androgenetic males, readily identifiable by their recessive paternal traits. The androgenetic males were carefully inspected for visible mutations.

Due to the fact that these males are haploid, both dominant and recessive mutations would be apparent in them. The biparental females were set unmated and their haploid sons studied for visible mutations. These females, half-sisters of androgenetic males, would be heterozygous for any visible mutations induced in the x-rayed egg chromosomes since they had developed from one set of x-rayed and one set of untreated chromosomes. The irradiated eggs were fertilized at times varying from one to several hours after exposure.

During the course of the experiments 6714 wild-type females were x-rayed, 2414 F_1 females tested and 75,546 F_2 males counted in the search for visible mutations (table 1). The number of androgenetic males at any one dose is too small to be significantly compared with their half-sisters

TABLE 1

DOSE IN r UNITS	VISIBLE MUTATIONS HETEROZYGOUS ♀ ♀ ♀ ♀ TESTED	%	ANDROGENETIC ♂ ♂
1,350	1/111	0.90	1
2,025	1/246	0.40	4
6,000	1/285	0.35	7
7,210	1/54	1.85	0
12,000	30/585	5.13	13
14,420	9/139	6.47	22
21,630	31/422	7.34	31
28,840	18/223	8.07	39
34,608	....	...	9
36,050	37/324	11.42	24
40,000	....	...	3
43,260	3/25	12.00	4
Miscellaneous	....	...	13
	132/2414 = 5.470%		170

2P = 0.0002 (obtained by exact method of treating contingency tables).

in respect to visible mutation rate. A comparison of totals, 0/170[9] or 0% mutation rate for androgenetic males and 132/2414 or 5.470% for their half-sisters, indicates that the difference is highly significant. The exact method of treating contingency tables was used and 2 P was found to be 0.0002.

Androgenetic males of Habrobracon owe their *origin* to injury of irradiated egg *chromosomes*, their *survival* to absence of injury in the x-rayed egg *cytoplasm*.[10, 11] If these were the only factors involved in the incidence of androgenetic males, however, they would increase with increase in dose until the limit for their production was reached, the number of fertilized eggs capable of developing into them. Maximum number of such eggs per female is about thirteen. Maximum number of androgenetic males per female is about 0.12. This is attained at the relatively low dose of about 15,000 r. At higher doses a factor which reduces androgenetic males begins

to take effect and they decrease in number until none appears at about 54,000 r.[12] They have the same dose limit for survival as do gynogenetic males and biparental females. The single factor which all these classes of progeny have in common is x-rayed cytoplasm. Cytoplasm can, therefore, be injured by x-rays. Nevertheless it has exhibited no mutagenic effect. Of androgenetic males studied in the present work, 123 or 72.3% developed in cytoplasm which had been treated with doses high enough to induce detectable cytoplasmic injury. In spite of this they were normal in appearance and all tested were found to be fully fertile.

A question might be raised concerning the relative sensitivity to x-rays of the mitotic stages of the nuclei under discussion. The irradiated nuclei taking part in the formation of biparental females were treated in the diffuse stage of the first meiotic prophase. Sperm chromosomes become diffuse very soon after entering the egg. The chromosomes do not differ strikingly, either in degree of condensation or in tension, therefore, at times of exposure to possible mutagenic influences.

All results obtained from the study of the responses of Habrobracon eggs to x-rays are consistent in their indication that two kinds of change may be induced in the cell: (1) chromosome alterations connected with the production of dominant and recessive lethal and visible mutations and (2) a lethal effect directly cytoplasmic. The former are induced directly in the chromosomes or by transitory effect on the cytoplasm and have no lower threshold. The dose limits above which there is no survival may vary greatly in different mitotic stages of the same kind of cell. The latter has a high threshold and could not be detected in any cell with sensitive chromosomes except by such a method as the one used in this study. In the Habrobracon egg this cytoplasmic injury is constant in respect to dose for its incidence and dose for complete lethal action, irrespective of stage of the chromosomes at time of treatment.[10-12]

It is possible that mutagenic changes in chromosomes are induced by temporary alteration of irradiated cytoplasm since the shortest period between exposure of the egg and fertilization in the present study was one hour. This allowed time for recovery of cytoplasm before fertilization. If, however, there is such temporary cytoplasmic injury, there remains the conclusion, unavoidable to the author, that this is quite distinct from that induced by high doses which has been described by Duryee and Petrova.

The kinds of change with which the cytogeneticist works, "point" mutations, inversions, translocations, deletions, could not be identified in Duryee's material. These changes may have been induced in nuclei x-rayed after removal from the cytoplasm. The fact that such nuclei do not appear to have reacted markedly does not rule out mutational effects. The significance of the changes observed by Duryee in the three sets of eggs, nucleus x-rayed, cytoplasm x-rayed and both x-rayed, can be deter-

mined with certainty only if progeny can be obtained from them or if detailed analysis of chromosome behavior in subsequent meiotic divisions can be made. No author has reported induction of visible mutations by means of irradiation of the cytoplasm alone.

Conclusion.—X-rays can induce permanent change in the cytoplasm of Habrobracon eggs. This change may have a lethal effect on the egg but has not induced visible mutations in untreated chromosomes.

* This investigation was completed with the aid of a research grant from the National Cancer Institute of the National Institutes of Health, U. S. Public Health Service. The work was done at the Zoological Laboratory of the University of Pennsylvania and at the Marine Biological Laboratory, Woods Hole, Massachusetts. Valuable assistance was given the author by Bernice Goldis and Constance Mitchell.

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[7] Duryee, W. R., *Anat. Record*, **75**, 144 (1939).

[8] Duryee, W. R., *Biol. Bull.*, **93**, 206 (1947).

[9] Since this paper was written the students in the Genetics Seminar at Swarthmore College have obtained twenty-nine additional androgenetic males. All were normal in appearance. Dose used was 28,000 r.

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A NOTE ON THE EXCEPTIONAL JORDAN ALGEBRA

By A. A. Albert

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Communicated May 29, 1950

An associative algebra $\mathfrak{A}$ over a field $\mathfrak{F}$ is a vector space over $\mathfrak{F}$ together with an associative bilinear operation xy. When the characteristic of $\mathfrak{F}$ is not two we can use the same vector space and define a new algebra $\mathfrak{A}^{(+)}$ relative to the operation $\frac{1}{2}(xy + yx)$. This algebra is a Jordan algebra. Any Jordan algebra $\mathfrak{J}$ is called a *special* Jordan algebra if $\mathfrak{J}$ is isomorphic to a Jordan subalgebra of some $\mathfrak{A}^{(+)}$.

In 1934 it was shown[1] that the Jordan algebra $\mathfrak{H}$ of all three-rowed Hermitian matrices with elements in the simple eight-dimensional Cayley algebra $\mathfrak{C}$ is *exceptional* in the limited sense that $\mathfrak{H}$ is not isomorphic to a subalgebra of a finite-dimensional $\mathfrak{A}^{(+)}$. In the present note we shall give a simpler proof of the fact that $\mathfrak{H}$ is not a special Jordan algebra and *shall*

delete the restriction that $\mathfrak{A}$ be finite dimensional. We shall assume that $\mathfrak{G}$ is imbedded in an associative algebra $\mathfrak{A}$ and shall then obtain a contradiction by showing that the enveloping associative algebra of $\mathfrak{G}$ contains a subalgebra isomorphic to the non-associative algebra $\mathfrak{C}$.

We shall begin with the following background material. Let the product in $\mathfrak{C}$ be designated by $x \cdot y$ and the involution of $\mathfrak{C}$ by $x \to \bar{x}$, and let u be the unity quantity of $\mathfrak{C}$. The algebra $\mathfrak{G}$ is the supplementary sum $\mathfrak{G} = \mathfrak{G}_{11} + \mathfrak{G}_{22} + \mathfrak{G}_{33} + \mathfrak{G}_{12} + \mathfrak{G}_{13} + \mathfrak{G}_{23}$ of vector spaces $\mathfrak{G}_{ij}$ for $i \leqq j$ where $\mathfrak{G}_{ii} = e_i\mathfrak{F}$ for pairwise orthogonal idempotents e_i whose sum is the unity quantity of $\mathfrak{G}$. The spaces $\mathfrak{G}_{12}$, $\mathfrak{G}_{13}$, $\mathfrak{G}_{23}$ are isomorphic in the vector space sense to $\mathfrak{C}$ and we express this isomorphism by writing x_{ij} for the general quantity of $\mathfrak{G}_{ij}$ for every $i < j$, where x is the general quantity of $\mathfrak{C}$. Then u defines a quantity u_{ij} such that

$$u_{ij}^2 = e_i + e_j. \tag{1}$$

The properties of $\mathfrak{G}$ may now be expressed in terms of the operation xy of $\mathfrak{A}$ and we have

$$e_ix_{ij} + x_{ij}e_i = e_jx_{ij} + x_{ij}e_j = x_{ij},\ e_kx_{ij} + x_{ij}e_k = 0 \tag{2}$$

for $i < j$ and $k \neq i, j$. We also have[2] the properties

$$\begin{gathered} x_{12}y_{23} + y_{23}x_{12} = (x \cdot y)_{13},\ x_{12}y_{13} + y_{13}x_{12} = (\bar{x} \cdot y)_{23}, \\ x_{12}y_{23} + y_{23}x_{12} = (x \cdot \bar{y})_{12}. \end{gathered} \tag{3}$$

Let us now proceed to our proof.

We first see that $0 = e_k(e_kx_{ij} + x_{ij}e_k) = (e_kx_{ij} + x_{ij}e_k)e_k$. Since $e_k^2 = e_k$ we have $e_kx_{ij} = x_{ij}e_k$ and so $2e_kx_{ij} = 0$,

$$e_kx_{ij} = x_{ij}e_k = 0. \tag{4}$$

Write $f = e_i + e_j$ so that f is an idempotent and $fx_{ij} + x_{ij}f = 2x_{ij}$. Then $2fx_{ij} = fx_{ij} + fx_{ij}f$, $fx_{ij} = fx_{ij}f = x_{ij}f$ and so

$$x_{ij} = x_{ij}(e_i + e_j) = (e_i + e_j)x_{ij}. \tag{5}$$

We finally see that $e_iy_{ij}e_i = e_i(y_{ij} - e_iy_{ij}) = e_iy_{ij} - e_iy_{ij} = 0$ and so we have

$$e_iy_{ij}e_i = e_jy_{ij}e_j = 0. \tag{6}$$

Define

$$x' = e_1x_{12}u_{12}$$

for every x of $\mathfrak{C}$. Then $(x \cdot y)' = e_1(x \cdot y)_{12}u_{12} = e_1(x_{12}\bar{y}_{23} + \bar{y}_{23}x_{13})u_{12} = e_1x_{13}\bar{y}_{23}u_{12}$ by (3) and (4). Use the first equation of (3) with $y = u$, $x \cdot y = x$ to obtain $x_{13} = x_{12}u_{23} + u_{23}x_{12}$ whence $(x \cdot y)' = e_1(x_{12}u_{23} + u_{23}x_{12})\bar{y}_{23}u_{12} = e_1x_{12}u_{23}\bar{y}_{23}u_{12}$. By the second equation of (3) we have $\bar{y}_{23} = y_{12}u_{13} + u_{13}y_{12}$

and so $(x\cdot y)' = e_1x_{12}u_{22}(y_{12}u_{13} + u_{12}y_{12})u_{12}$. Now (4), (5) and (6) imply that

$$y_{12}u_{13}u_{12} = y_{12}u_{13}(e_1 + e_2)u_{12} = (\mathfrak{H}_{23} - u_{13}y_{12})e_1u_{12} = -u_{13}y_{12}e_1u_{12} =$$

$$-u_{12}(e_1 + e_2)y_{12}e_1y_{12} = -u_{12}e_1y_{12}e_1u_{12} = 0.$$

Also $u_{23}u_{13}y_{12} = u_{23}u_{13}(e_1 + e_2)y_{12} = u_{23}u_{12}e_1y_{12} = (u_{12} - u_{13}u_{23})e_1y_{12} = u_{12}e_1y_{12}$. But then $(x\cdot y)' = e_1x_{12}u_{12}e_1y_{12}u_{12} = x'y'$.

We have proved that the mapping $x \to x'$ is a homomorphism of $\mathfrak{C}$ onto the subalgebra $\mathfrak{C}'$ of $\mathfrak{A}$ consisting of all x'. The kernel $\mathfrak{H}$ of this homomorphism is not $\mathfrak{C}$ since otherwise every $x' = 0$ whereas $u' = e_1u_{12}^2 = e_1(e_1 + e_2) = e_1 \neq 0$. Since $\mathfrak{C}$ is simple $\mathfrak{H} = 0$, the homomorphism is an isomorphism. This is impossible since $\mathfrak{C}$ is not associative and $\mathfrak{C}'$ is associative.

[1] See the author's "On a Certain Algebra of Quantum Mechanics," *Ann. Math.*, **35**, 65–73 (1934).

[2] For these properties see Section 18 of the author's "A Structure Theory for Jordan Algebras," *Ibid.*, **48**, 546–567 (1947).

ON THE SINGULAR VALUES OF A PRODUCT OF COMPLETELY CONTINUOUS OPERATORS

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Communicated by G. C. Evans, May 16, 1950

In this note I wish to present a theorem on the singular values of a product of completely continuous operators in Hilbert space. As an application, a simple proof of a recent result of S. H. Chang will be given. The singular values of an operator K are the positive square roots of the eigen-values of K^*K, where K^* is the adjoint of K.

We begin with a slight generalization of a theorem of Weyl.[1]

THEOREM 1. *If H is a positive, symmetric, completely continuous operator whose first n eigen-values[2] are $\lambda_1, \ldots, \lambda_n$, then*

$$det\ [(Hy_i, y_j)] \leq \lambda_1 \cdot \ldots \cdot \lambda_n\ det\ [(y_i, y_j)]$$

for any elements $y_1, \ldots, y_n$.

Here, det $[a_{ij}]$ denotes the determinant of the nth order matrix with elements a_{ij}. Weyl's elegant proof uses an appeal to the theory of n-tensors. A straightforward proof may be given by using the relation $(Hy_i, y_j) = \sum_k \lambda_k (y_i, x_k)(x_k, y_j)$, where the x_k form a *complete* ortho-normal set.

THEOREM 2. *If K is a completely continuous operator with singular values α_i, then det $[(Ky_i, Ky_j)] \leq \alpha_1^2 \ldots \alpha_n^2$ det $[(y_i, y_j)]$.*

This follows immediately from Theorem 1 if we set $H = K^*K$.

THEOREM 3. *Let A and B be completely continuous operators and let the singular values of A, B and AB be denoted by α_i, β_i, γ_i, respectively. If f is any function such that $f(e^x)$ is convex and increasing as a function of x, then for each n we have $\sum_{i=1}^{n} f(\gamma_i) \leq \sum_{i=1}^{n} f(\alpha_i\beta_i)$.*

Proof: Let $y_1, \ldots, y_n$ form an ortho-normal set with $(AB)^*ABy_i = \gamma_i^2 y_i$. By Theorem 2, $\gamma_1^2 \cdot \ldots \cdot \gamma_n^2 = \det [(ABy_i, ABy_j)] \leq \alpha_1^2 \cdot \ldots \cdot \alpha_n^2$ det $[(By_i, By_j)] \leq \alpha_1^2 \cdot \ldots \cdot \alpha_n^2 \beta_1^2 \cdot \ldots \cdot \beta_n^2$. The result now follows by an application of a theorem of Polya.[3]

The next theorem was proved by Chang[4] using methods of function theory.

THEOREM 4. *Suppose $K = K_1 \cdot \ldots \cdot K_m$, where each K_i is an operator of finite norm (integral operator with L_2 kernel), and let γ_i be the singular values of K. Then $\sum_i \gamma_i^{2/m}$ converges.*

Proof: The proof is by induction on m. The case $m = 1$ is an immediate consequence of the definition of an operator of finite norm. Suppose the theorem holds when K is a product of fewer than m operators. Let α_i be the singular values of K_1, and let β_i be the singular values of $K_2 \cdot \ldots \cdot K_m$. Using Theorem 3 and Holder's inequality, we have

$$\sum_i \gamma_i^{2/m} \leq \sum_i \alpha_i^{2/m} \beta_i^{2/m} \leq \left(\sum_i \alpha_i^2\right)^{1/m} \left(\sum_i \beta_i^{2/m-1}\right)^{m-1/m}$$

In conclusion we remark that by a theorem of Chang,[4] the convergence of $\sum_i \gamma_i^{2/m}$ implies the convergence of $\sum_i |\lambda_i|^{2/m}$, where λ_i are the eigenvalues of K.

[1] Weyl, H., "Inequalities Between the Two Kinds of Eigenvalues of a Linear Transformation," these PROCEEDINGS, **35**, 408–411 (1949).

[2] The eigen-values and singular values will always be arranged in order of decreasing absolute value, with repetitions according to multiplicity.

[3] Polya, G., "Remark on Weyl's Note: Inequalities Between the Two Kinds of Eigenvalues of a Linear Transformation," these PROCEEDINGS, **36**, 49–51 (1950).

[4] Chang, S. H., "On the Distribution of the Characteristic Values and Singular Values of Linear Integral Equations," *Trans. Am. Math. Soc.*, **67**, 351–368 (1949).

SOME NEW FUNCTIONS OF INTEREST IN X-RAY CRYSTALLOGRAPHY*

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Communicated by W. J. Mead, May 15, 1950

In an earlier contribution[1] the relations between a *fundamental set* of points and its *vector set* were discussed, and it was shown that there are several ways of deriving the fundamental set from its vector set. The theory was confined to a discussion of vector sets based on fundamental sets composed of discrete unit points.

The theory can be readily extended to sets of weighted points and to density maps. Let fundamental space be divided into "unit" volumes. In each volume there are a number of points (which may be zero). Consider the particular "unit" volumes surrounding points a and b. Let the number of points in a's volume be m, and the number of points in b's volume be n. There are mn vectors from the points in a's volume to the points in b's volume; and there are nm vectors from the points in b's volume to the points in a's volume. If these vectors are now transferred to the common origin in vector space, they become two centrosymmetrical sheaves of vectors. The number of points at the end of each sheaf is mn. If the "unit" volumes are now reduced to zero, the m points in the volume at a come to coincide with point a, and the n points in the volume at b come to coincide with point b. In the vector set, the mn points in the volume at ab come to coincide with vector point ab. Since a is always associated with m, and b with n, the designations a and b may be used not only to indicate the points a and b but also may be permitted to carry the connotation of the number of points coincident at a and b, namely the weights of points a and b.

Now suppose that one decomposes a vector set into identical polygons.[1] For definiteness, suppose that the fundamental set is a five-point set, $a + b + c + d + e$, figure 1 (A). The decomposition is represented by equating the matrix of the vector set to the five images of the pentagon $a + b + c + d + e$, as follows:

$$\begin{matrix} aa & ab & ac & ad & ae \\ ba & bb & bc & bd & be \\ ca & cb & cc & cd & ce \\ da & db & dc & dd & de \\ ea & eb & ec & ed & ee \end{matrix} = \begin{matrix} a(a+b+c+d+e) \\ b(a+b+c+d+e) \\ c(a+b+c+d+e) \\ d(a+b+c+d+e) \\ e(a+b+c+d+e) \end{matrix} \qquad (1)$$

This decomposition demonstrates that, in general, each of the polygons in the vector set has a different weight, the various weights being the coeffi-

cients of the polynomials on the right of (1). As a consequence, the several different solutions of a vector set appear differently weighted, as diagrammatically indicated in figure 1 (*B*). If the weighting in the fundamental set is taken as the numbers of electrons in point-atoms, then the weighting in the vector set is that of the weights of the point-atoms in the squared-crystal.[2] If the weighting in the fundamental set is taken as the electron density, then the corresponding weighting in the vector set maps out the Patterson function.

The connection between vector maps of discrete points and the Patterson map can be traced as follows: Divide fundamental space and vector space

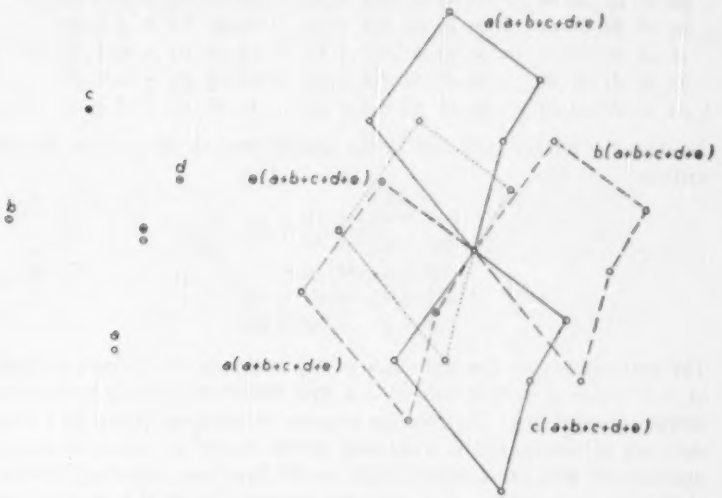

FIGURE 1A FIGURE 1B

into elementary volumes, Δv. If the density of points at positions a and b in fundamental space are ρ_a and ρ_b, respectively, then there are $\rho_a \Delta v$ and $\rho_b \Delta v$ points in the elementary areas containing positions a and b. At the position ab in vector space there are $\rho_a \Delta v \times \rho_b \Delta v$ points in a volume $8\Delta v$. The density of points is therefore $\rho_a \Delta v \times \rho_b \Delta v / 8\Delta v = {}^1/_8 \rho_a \rho_b \Delta v$. Neighboring cells of volume $8\Delta v$ overlap one another in such a way that each makes density contributions to neighboring cells. When this is taken into account, the actual density at point ab is $\rho_a \rho_b \Delta v$. As $\Delta v \to dv$, the value of the vector set weight at a particular point ab approaches $\rho_a \rho_b dv$. If there are other pairs of points in fundamental space parallel to $\overrightarrow{ab}$ which both have non-zero weights, then the weighting at ab in vector space is $\int_0^V \rho_a \rho_b dv$.

In the Patterson function, the average value of this product over the volume of the cell is chosen, namely $\frac{1}{V}\int_0^V \rho_u \rho_v dv$. Thus, the vector set of an electron density map has values at each point proportional to the Patterson function at these points.

For purposes to appear later, attention is next directed to the geometrical relation between the set of images of any particular polygon in the vector set. Let the vector set matrix be separated into images of a line, of a triangle, ... of an n-gon, as follows:

$$\begin{array}{ccccc} aa & ab & ac & ad & ae \\ ba & bb & bc & bd & be \\ ca & cb & cc & cd & ce \\ da & db & dc & dd & de \\ ea & eb & ec & ed & ee \end{array} = \begin{array}{cccc} aa & ab & ac & a(d+e) \\ ba & bb & bc & b(d+e) \\ ca & cb & cc & c(d+e) \\ da & db & dc & d(d+e) \\ ea & eb & ec & e(d+e) \end{array} = \begin{array}{ccc} aa & ab & a(c+d+e) \\ ba & bb & b(c+d+e) \\ ca & cb & c(c+d+e) \\ da & db & d(c+d+e) \\ ea & eb & e(c+d+e) \end{array} \quad (2)$$

Consider the images indicated in the second part of (2). These can be written

$$\begin{array}{rcl} a(d+e) & = & ad + ae \\ b(d+e) & = & bd + be \\ c(d+e) & = & cd + ce. \\ d(d+e) & = & dd + de \\ e(d+e) & = & ed + ee \end{array} \quad (3)$$

The first column on the right side of (3) is simply the column polygon $(a + b + c + d + e)$ as imaged in d, and the second column is the same polygon imaged in e. In a similar manner, homologous points in a complete set of row images is a solution of the vector set which is centrosymmetrical with the solution which would have been achieved by completing any original row image. The centrosymmetrical aspect follows because corresponding row and column images are centrosymmetrical.

Sufficient groundwork is now established for considering a function which has the property of transforming a Patterson map into a representation of the electron density. If one could devise a function which would seek points located at one end of a line image, this collection of points would correspond to a column of the right of (3) and hence would be a weighted solution of the vector set. A function which has a high value when a line, whose components are xyz, spans two Patterson peaks is the product of the two Patterson functions at the ends of the line, namely

$$\mathrm{II}_2(uvw, xyz) = P(uvw) \times P(u + x, v + y, w + z). \quad (4)$$

Such a function has maxima for values of uvw corresponding to all points which occur at the end of a line image of components xyz.

In using the function, it is undesirable to let x, y and z vary through their

entire range. If this were permitted, every point in the vector set would be reproduced by the function, for *every* vector point is related to other vector points by line images. Rather, it is desirable to set up the function to some particular line image. To accomplish this one first finds, in the Patterson map, a point image which is single. If this point has coordinates $x_1y_1z_1$, then the line from the origin to this point is one of a family of line images. The components of the line are $x_1y_1z_1$. If this specific value is placed in (4), then the function seeks the other points of the map which are at corresponding points of the line images of the rest of this family.

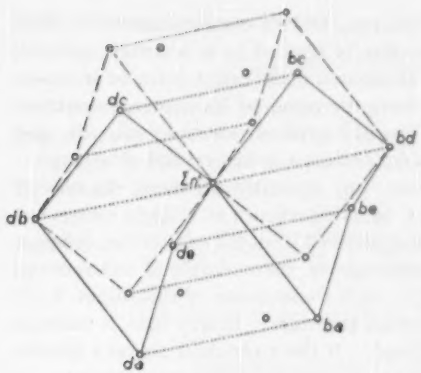

FIGURE 2A

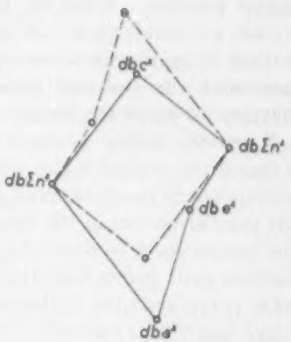

FIGURE 2B

A more symmetrical form of this function may be arranged by requiring a high value of the function at a point at the center of gravity of the image. For line images, such a symmetrical function has the form

$$\Pi_2(uvw, xyz) = P\left(u - \frac{x}{2}, v - \frac{y}{2}, w - \frac{z}{2}\right) \times P\left(u + \frac{x}{2}, v + \frac{y}{2}, 2 + \frac{z}{2}\right). \quad (5)$$

Some of the characteristics of this function are illustrated in figure 2. Figure 2 (*A*) shows the vector set of figure 1 (*B*). Before the function is set up, one first makes the decision that some point in the vector set, namely *bd* in figure 2 (*A*), represents the image of a single point. The dotted line from the origin to this point is the line image which is thus established, and function (5) is then set up for the components of this line. The function has a value equal to the product of the values at the ends of this line as the line is shifted parallel to itself so as to range over the entire volume of the vector set. In the vector set of discrete points used for

illustration in figure 2 (*A*), the vector set has non-zero values only at the points shown by small circles. Wherever the image line spans a pair of points, the function lays down a peak at the center of the line. The map of the function and its values are shown in figure 2 (*B*). Note that the weight of a point in the function is the square of the weight in the fundamental set (Fig. 1 (*A*)) times a scale factor, *db*, with the proviso that points corresponding to images containing the origin are distorted to the origin weight of the vector set. The scale factor is the product used as the image point, namely *bd*. Thus, function (5) maps the electron density from Patterson F^2 data, except that it maps the density as its square, and that it maps the original image atom as having a weight equal to the Patterson origin weight.

Figure 2 (*B*) also shows that this map and its centrosymmetrical ghost appear together. When the function is applied to a centrosymmetrical crystal, a separate ghost can be eliminated if the origin point *bd* is chosen so that it represents a vector from an atom to its centrosymmetrical equivalent. In this case these enantiomorphous solutions coincide, and function (5) maps the desired atom positions in the crystal structure.

A proviso, tacitly assumed here, but explicitly discussed elsewhere,* is that if the original image point, *bd*, is not chosen at a single image, but corresponds to two coincident points derived from the ends of two different but parallel vectors in the fundamental set, then additional and spurious line images occur in figure 2 (*A*). As a consequence of this, figure 2 (*B*) contains more points than the crystal structure. In any case, it contains fewer points than the Patterson map. If the x-ray data are on a quantitative basis, it is currently possible to identify single image points. A convenient way to locate desirable image points is to use appropriate peaks in a Harker section.

Function (5) can be computed in either of two ways: If the numerical values are available for the Patterson function at an array of points *uvw*, which is usual after computing the function by using the Beevers-Lipson method[3] or some variation of it, then the Patterson function values separated by a line with components *xyz* can be multiplied to produce (5) at the midpoint of the line. This ordinarily requires 3600 products for a two-dimensional map unless the crystal has some symmetry, when this number is correspondingly less. Alternatively (5) can be recast into a Fourier synthesis based on the original F^2's. The form of the computation is found by substituting in (5) the values of the Patterson function, namely

$$P(uvw) = \frac{1}{V^2} \sum_h \sum_k \sum_l F^2_{hkl}\, e^{2\pi i(hu + kv + lw)}. \qquad (6)$$

This gives

$$\Pi_2(uvw, xyz) = \frac{1}{V^2} \sum_{h_1} \sum_{k_1} \sum_{l_1} F^2_{h_1 k_1 l_1}\, e^{2\pi i(h_1[u - (x/2)] + k_1[v - (y/2)] + l_1[w - (z/2)])} \times$$

$$\frac{1}{V^2}\sum_{h_2}\sum_{k_2}\sum_{l_2} F^2_{h_2k_2l_2}\, e^{2\pi i(h_2[u+(x/2)]+k_2[v+(y/2)]+l_2[w+(z/2)])} \quad (7)$$

$$= \frac{1}{V^4}\sum_{h_1}\sum_{h_2}\sum_{k_1}\sum_{k_2}\sum_{l_1}\sum_{l_2} F^2_{h_1k_1l_1}F^2_{h_2k_2l_2} \times$$
$$e^{2\pi i([h_1-h_2]x/2+[k_1-k_2]y/2+[l_1-l_2]z/2} \times$$
$$e^{2\pi i([h_1+h_2]u+[k_1+k_2]v+[l_1+l_2]w)}. \quad (8)$$

This can be simplified for the usual experimental conditions, under which Friedel's law[4]

$$F^2_{hkl} = F^2_{\bar{h}\bar{k}\bar{l}} \quad (9)$$

holds. Under these conditions,

$$F^2_{h_1k_1l_1}F^2_{h_2k_2l_2} = F^2_{\bar{h}_1\bar{k}_1\bar{l}_1}F^2_{\bar{h}_2\bar{k}_2\bar{l}_2}, \text{ etc.,} \quad (10)$$

and the exponentials accompanying these coefficients in (8) determine opposite phases. In the entire summation, therefore, the imaginary components of the exponentials vanish, leaving only their real components, so that (8) can be rewritten

$$\Pi_2(uvw, xyz) = \frac{1}{V^4}\sum_{h_1}\sum_{h_2}\sum_{k_1}\sum_{k_2}\sum_{l_1}\sum_{l_2} F^2_{h_1k_1l_1}F^2_{h_2k_2l_2} \times$$
$$\cos 2\pi([h_1-h_2]\frac{x}{2}+[k_1-k_2]\frac{y}{2}+[l_1-l_2]\frac{z}{2}) \times$$
$$\cos 2\pi([h_1+h_2]u+[k_1+k_2]v+[l_1+l_2]w). \quad (11)$$

Part of the indicated summations as well as the first cosine term are not concerned with the Fourier computation proper, but merely relate to the formation of the compound Fourier coefficients. Thus (11) is a Fourier series resembling the standard electron density summation.

The decomposition of the vector set matrix into images shown in (2) indicates that homologous points of any complete set of polygon images maps out the fundamental set. Therefore, in a manner analogous to devising a function for finding line images, a function can be designed to find homologous points associated with any image. Suppose one has a Patterson map on which two peaks can be distinguished which (one has reason to believe) are both images of single points. Let their coordinates be $x_1y_1z_1$ and $x_2y_2z_2$. These two peaks and the origin constitute the image of a triangle with a point at the origin. For this triangle and all of the other images of the triangle, the function

$$\Pi_3(uvw, x_1y_1z_1, x_2y_2z_2) = P(uvw) \times P(u+x_1, v+y_1, w+z_1) \times$$
$$P(u+x_2, v+y_2, w+z_2) \quad (12)$$

has a high value. Therefore this function maps out the location of the atoms in the crystal structure. The value of this function at each point in the structure is the cube of the electron density, times a scale factor depending on the image points chosen, except that the density at the points corresponding to the origin triangles is exaggerated. (The form of (12), which corresponds with the form of (4) can also be expressed in a more symmetrical form corresponding with that of (5).)

In a similar manner more complicated image-seeking functions, Π_n, which seek images of a chosen n-gon, can be set up. The greater the value of n, the greater the distortion of the electron density mapped by the function, and the more tedious the computation, but the more certainly does the function reproduce variations in electron density rather than yield to fortuitous coincidences. This is because the function requires cooperation between n Patterson peaks to produce one electron density peak.

Since Π_2 maps the square of the electron density, Π_3 maps the cube of the electron density, etc., it follows that $\sqrt[2]{\Pi_2}$, $\sqrt[3]{\Pi_3}$, ... $\sqrt[n]{\Pi_n}$ map the electron density, except that atoms derived from images in the Patterson which contain the origin are exaggerated. It also follows that the real parts of the phases of the Fourier coefficients of the electron density function are the same as phases of the functions $\sqrt[2]{\Pi_2}$, $\sqrt[3]{\Pi_3}$ ... $\sqrt[n]{\Pi_n}$ except that an allowance must be made for the exaggeration of the atoms arising from the origin image.

The discussion has been carried out largely in terms of three-dimensional Patterson functions. The successful use of Patterson functions for anything but simple structures depends largely on the use of three-dimensional functions, since two-dimensional and one-dimensional functions show bad overlapping of projected peaks.

The image-seeking function (11) has a resemblance to a function recently described by McLachlan.[5] The function given here differs in method of derivation and in the details of its form, specifically in the form of the Fourier coefficients.

[*] The subject matter of this paper formed part of a larger contribution entitled, "The Application of Image Theory to Crystal Structure Analysis," presented at the Phase and Computor Conference, Pennsylvania State College, April 6, 1950.

[1] Buerger, M. J., "Vector Sets," *Acta Cryst.*, **3**, 87–97 (1950).

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[4] Friedel, G., "Sur les symetries cristallines que peut reveler la diffraction des rayons Röntgen," *Compt. Rend., Paris*, **157**, 1533–1536 (1913).

[5] McLachlan, Dan, Jr., "The Use of Mixed Projections in the Solution of Crystal Structures." (Paper read before the meeting of the Am. Cryst. Assoc., Penn State College, April 10, 1950.)

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Bainbridge, Kenneth Tompkins, 1946 (3), Department of Physics, Harvard University, Cambridge 38, Mass.

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Bancroft, Wilder Dwight, 1920 (5), 7 East Avenue, Ithaca, N. Y.

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Beams, Jesse **Wakefield,** 1943 (3), Rouss Physical Laboratory, University of Virginia, Charlottesville, Va.

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Birge, Raymond Thayer, 1932 (3), University of California, Berkeley 4, Calif.

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Blackwelder, Eliot, 1936 (6), P. O. Box N, Stanford University, Calif.

Blake, Francis Gilman, 1947 (10), Yale University School of Medicine, 333 Cedar Street, New Haven 11, Conn.

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Briggs, Lyman James, 1942 (4), National Bureau of Standards, Washington 25, D. C.

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Bronk, Detlev Wulf, 1939 (9), Johns Hopkins University, Baltimore 18, Md.

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Castle, William Bosworth, 1939 (10), Boston City Hospital, Boston 18, Mass.

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Coggeshall, Lowell Thelwell, 1949 (10), Division of Biological Sciences, University of Chicago, Chicago 37, Ill.

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Hutchinson, George Evelyn, 1950 (8), Osborn Zoological Laboratory, Yale University, New Haven 11, Conn.

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Lewis, Warren Kendall, 1938 (4), Massachusetts Institute of Technology, Cambridge 39, Mass.

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Loomis, Alfred Lee, 1941 (4), The Loomis Laboratory, Room 2420, 14 Wall St., New York 5, N. Y.

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Mann, Frank Charles, 1950 (9), Mayo Foundation, University of Minnesota, Rochester, Minn.

Marshall, Eli Kennerly, Jr., 1943 (9), School of Medicine, Johns Hopkins University, 710 North Washington Street, Baltimore 5, Md.

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Nicholas, John Spangler, 1949 (8), Osborn Zoological Laboratory, Yale University, New Haven 11, Conn.

Nicholson, Seth Barnes, 1937 (2), Mount Wilson Observatory, Pasadena 4, Calif.

Nier, Alfred Otto C., 1950 (3), Department of Physics, University of Minnesota, Minneapolis 14, Minn.

Northrop, John Howard, 1934 (9), Department of Bacteriology, University of California, Berkeley 4, Calif.

Novy, Frederick George, 1924 (10), 721 Forest Avenue, Ann Arbor, Mich.

Noyes, William Albert, Jr., 1943 (5), Department of Chemistry, University of Rochester, Rochester 3, N. Y.

Oncley, John Lawrence, 1947 (9), University Laboratory of Physical Chemistry, Harvard University, 25 Shattuck St., Boston 15, Mass.

Onsager, Lars, 1947 (5), Sterling Chemistry Laboratory, Yale University, 225 Prospect Street, New Haven, Conn.

Opie, Eugene Lindsay, 1923 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, N. Y.

Oppenheimer, J. Robert, 1941 (3), The Institute for Advanced Study, Princeton, N. J.

Osterhout, Winthrop John Vanleuven, 1919 (7), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, N. Y.

Painter, Theophilus Shickel, 1938 (8), University of Texas, Austin 12, Tex.

Palache, Charles, 1934 (6), Harvard University, Cambridge 38, Mass.

Parker, George Howard, 1913 (8), Harvard Biological Laboratories, Divinity Avenue, Cambridge 38, Mass.

Patterson, John Thomas, 1941 (8), Department of Zoology, University of Texas, Austin 12, Tex.

Paul, John Rodman, 1945 (10), Yale University School of Medicine, 333 Cedar Street, New Haven 11, Conn.

Pauling, Linus, 1933 (5), Crellin Laboratory, California Institute of Technology, Pasadena 4, Calif.

Pegram, George Braxton, 1949 (3), Columbia University, New York 27, N. Y.

Peters, John Punnett, 1947 (9), Department of Internal Medicine, Yale University School of Medicine, New Haven 11, Conn.

Pierce, George Washington, 1920 (3), Cruft Laboratory, Harvard University, Cambridge 38, Mass.

Piggot, Charles Snowden, 1946 (3), United States Embassy, London, England

Pillsbury, Walter Bowers, 1925 (12), University of Michigan, Ann Arbor, Mich.

Pitzer, Kenneth Sanborn, 1949 (5), Division of Research, U. S. Atomic Energy Commission, 1901 Constitution Avenue, Washington 25, D. C.

Rabi, Isidor Isaac, 1940 (3), Department of Physics, Columbia University, New York 27, N. Y.

Raper, Kenneth Bryan, 1949 (7), Northern Regional Research Laboratory, U. S. Department of Agriculture, Peoria 5, Ill.

Raymond, Arthur Emmons, 1950 (4), Douglas Aircraft Company, Inc., 3000 Ocean Park Boulevard, Santa Monica, Calif.

Reeside, John Bernard, Jr., 1945 (6), Box 177, Hyattsville, Md.
Reichelderfer, Francis Wilton, 1945 (3), Room 310, U. S. Weather Bureau, Washington 25, D. C.
Rhoades, Marcus Morton, 1946 (7), Department of Botany, University of Illinois, Urbana, Ill.
Richards, Alfred Newton, 1927 (9), School of Medicine, University of Pennsylvania, Philadelphia 4, Pa.
Richter, Curt Paul, 1948 (12), The Johns Hopkins Hospital, Baltimore 5, Md.
Riddle, Oscar, 1939 (8), Route 4, Plant City, Fla.
Ritt, Joseph Fels, 1933 (1), Columbia University, New York 27, N. Y.
Rivers, Thomas Milton, 1934 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, N. Y.
Robbins, William Jacob, 1940 (7), New York Botanical Garden, Bronx Park (Fordham Station), New York 58, N. Y.
Robertson, Oswald Hope, 1943 (10), Department of Biology, Stanford University, Stanford University, Calif.
Rodebush, Worth Huff, 1938 (5), University of Illinois, Urbana, Ill.
Romer, Alfred Sherwood, 1944 (8), Museum of Comparative Zoology, Oxford Street, Cambridge 38, Mass.
Rose, William Cumming, 1936 (9), University of Illinois, Urbana, Ill.
Ross, Frank Elmore, 1930 (2), Mount Wilson Observatory, Pasadena 4, Calif.
Rossby, Carl-Gustaf Arvid, 1943 (3), Institute of Meteorology, University of Chicago, Chicago 37, Ill.
Rossi, Bruno Benedetto, 1950 (3), Department of Physics, Massachusetts Institute of Technology, Cambridge 39, Mass.
Rous, Francis Peyton, 1927 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, N. Y.
Rubey, William Walden, 1945 (6), United States Geological Survey, Washington 25, D. C.
Ruedemann, Rudolf, 1928 (6), 161 Dana Avenue, Albany 3, N. Y.
Russell, Henry Norris, 1918 (2), Princeton University, Princeton, N. J.
Sabin, Florence Rena, 1925 (10), 1333 East 10th Avenue, Denver 3, Colo.
Saunders, Frederick Albert, 1925 (3), South Hadley, Mass.
Savage, John Lucian, 1949 (4), 1651 Dahlia Street, Denver 7, Colo.
Sax, Karl, 1941 (7), Harvard University, Cambridge 38, Mass.
Scatchard, George, 1946 (5), Department of Chemistry, Massachusetts Institute of Technology, Cambridge 39, Mass.
Schlesinger, Hermann Irving, 1948 (5), Department of Chemistry, University of Chicago, Chicago 37, Ill.
Schmidt, Carl Frederic, 1949 (9), Laboratory of Pharmacology, School of Medicine, University of Pennsylvania, Philadelphia 4, Pa.
Schmitt, Francis Otto, 1948 (8), Department of Biology, Massachusetts Institute of Technology, Cambridge 39, Mass.
Schultz, Adolph Hans, 1939 (11), Johns Hopkins Medical School, Baltimore 5, Md.

Schwinger, Julian, 1949 (3), Department of Physics, Harvard University, Cambridge 38, Mass.

Seaborg, Glenn Theodore, 1948 (5), Department of Chemistry, University of California, Berkeley 4, Calif.

Seares, Frederick Hanley, 1919 (2), 351 Palmetto Drive, Pasadena 2, Calif.

Shaffer, Philip Anderson, 1928 (9), Washington University Medical School, St. Louis 10, Mo.

Shapiro, Harry Lionel, 1949 (11), Department of Anthropology, American Museum of Natural History, Central Park West at 79th Street, New York 24, N. Y.

Shapley, Harlow, 1924 (2), Harvard College Observatory, Cambridge 38, Mass.

Sherman, Henry Clapp, 1933 (9), Columbia University, New York 27, N. Y.

Shope, Richard Edwin, 1940 (10), Box 323, Kingston, N. J.

Simpson, George Gaylord, 1941 (6), American Museum of Natural History, 77th Street and Central Park West, New York 24, N. Y.

Sinnott, Edmund Ware, 1936 (7), Osborn Botanical Laboratory, Yale University, New Haven, Conn.

Skinner, Burrhus Frederic, 1950 (12), Psychological Laboratories, Harvard University, Cambridge 38, Mass.

Slater, John Clarke, 1932 (3), Massachusetts Institute of Technology, Cambridge 39, Mass.

Slepian, Joseph, 1941 (4), Westinghouse Electric Corporation, East Pittsburgh, Pa.

Slichter, Louis Byrne, 1944 (3), 1446 North Amalfi Drive, Pacific Palisades, Calif.

Slipher, Vesto Melvin, 1921 (2), Lowell Observatory, Flagstaff, Ariz.

Small, Lyndon Frederick, 1941 (5), National Institutes of Health, Bethesda 14, Md.

Smith, Gilbert Morgan, 1948 (7), School of Biological Sciences, Stanford University, Stanford University, Calif.

Smith, Homer William, 1945 (9), 477 First Avenue, New York 16, N. Y.

Smith, Lee Irvin, 1944 (5), School of Chemistry, University of Minnesota, Minneapolis 14, Minn.

Smith, Paul Althaus, 1947 (1) Department of Mathematics, Columbia University, New York 27, N. Y.

Smith, Philip Edward, 1939 (8), College of Physicians and Surgeons, 630 West 168th Street, New York 32, N. Y.

Soderberg, Carl Richard, 1947 (4), Massachusetts Institute of Technology, Cambridge 39, Mass.

Sonneborn, Tracy Morton, 1946 (8), Department of Zoology, Indiana University, Bloomington, Ind.

Spier, Leslie, 1946 (11), P. O. Box 880, Santa Cruz, Calif.

Stadie, William Christopher, 1945 (9), 821 Maloney Clinic, 36th and Spruce Streets, Philadelphia 4, Pa.

Stadler, Lewis John, 1938 (7), University of Missouri, Columbia, Mo.

Stakman, Elvin Charles, 1934 (7), University Farm, St. Paul 8, Minn.

Stanley, Wendell Meredith, 1941 (9), Department of Biochemistry, University of California, Berkeley 4, Calif.

Stebbins, Joel, 1920 (2), Lick Observatory, Mt. Hamilton, Calif.

Stern, Curt, 1948 (8), Department of Zoology, University of California, Berkeley 4, Calif.

Stern, Otto, 1945 (3), 759 Cragmont Avenue, Berkeley, Calif.

Stevens, Stanley Smith, 1946 (12), Department of Psychology, Harvard University, Cambridge 38, Mass.

Stewart, George Walter, 1938 (3), State University of Iowa, Iowa City, Iowa

Stock, Chester, 1948 (6), Division of Geological Sciences, California Institute of Technology, Pasadena 4, Calif.

Stone, Calvin Perry, 1943 (12), Stanford University, Stanford University, Calif.

Stone, Marshall Harvey, 1938 (1), 313 Eckhart Hall, University of Chicago, Chicago 37, Ill.

Stratton, Julius Adams, 1950 (4), Massachusetts Institute of Technology, Cambridge 39, Mass.

Struve, Otto, 1937 (2), Department of Astronomy, University of California, Berkeley 4, Calif.

Sturtevant, Alfred Henry, 1930 (8), California Institute of Technology, Pasadena 4, Calif.

Suits, Chauncey Guy, 1946 (4), Research Laboratory, General Electric Company, Schenectady 5, N. Y.

Sumner, James Batcheller, 1948 (9), New York State College of Agriculture, Laboratory of Enzyme Chemistry, Cornell University, Ithaca, N. Y.

Sverdrup, Harald Ulrik, 1945 (3), Norsk Polarinstitutt, Observatoriegt. 1, Oslo, Norway

Swanton, John Reed, 1932 (11), 22 George Street, Newton 58, Mass.

Taliaferro, William Hay, 1940 (8), University of Chicago, Chicago 37, Ill.

Teller, Edward, 1948 (3), P. O. Box 1663, Los Alamos, N. Mex.

Terman, Frederick Emmons, 1946 (4), School of Engineering, Stanford University, Stanford University, Calif.

Terman, Lewis Madison, 1928 (12), 761 Dolores Street, Stanford University, Calif.

Thimann, Kenneth Vivian, 1948 (7), Harvard University, Biological Laboratories, 16 Divinity Avenue, Cambridge 38, Mass.

Thom, Charles, 1937 (7), 207 Grant Street, Port Jefferson, N. Y.

Thomas, Charles Allen, 1948 (4), Monsanto Chemical Company, 1700 South Second Street, St. Louis 4, Mo.

Thomas, Tracy Yerkes, 1941 (1), Swain Hall, Indiana University, Bloomington, Ind.

Thurstone, Louis Leon, 1938 (12), University of Chicago, Chicago 37, Ill.

Timoshenko, Stephen Prokop, 1940 (4), Room 262, Engineering Building, Stanford University, Calif.

Tolman, Edward Chace, 1937 (12), Department of Psychology, University of California, Berkeley 4, Calif.

Tozzer, Alfred Marston, 1942 (11), Peabody Museum, Harvard University, Cambridge 38, Mass.

Trumpler, Robert Julius, 1932 (2), Berkeley Astronomical Department, University of California, Berkeley 4, Calif.

Tuve, Merle Antony, 1946 (3), Department of Terrestrial Magnetism, Carnegie Institution of Washington, 5241 Broad Branch Road, N. W., Washington 15, D. C.

Twitty, Victor Chandler, 1950 (8), Department of Biological Sciences, Stanford University, Stanford, Calif.

Tyzzer, Ernest Edward, 1942 (10), 175 Water Street, Wakefield, Mass.

Urey, Harold Clayton, 1935 (5), Institute for Nuclear Studies, University of Chicago, Chicago 37, Ill.

Vandiver, Harry Shultz, 1934 (1), Department of Applied Mathematics, University of Texas, Austin 12, Tex.

Van Niel, Cornelis Bernardus, 1945 (7), Hopkins Marine Station of Stanford University, Pacific Grove, Calif.

Van Slyke, Donald Dexter, 1921 (9), Brookhaven National Laboratory, Upton, Long Island, N. Y.

Van Vleck, John Hasbrouck, 1935 (3), Harvard University, Cambridge 38, Mass.

Vaughan, Thomas Wayland, 1921 (6), 3333 P Street, N. W., Washington 7, D. C.

Veblen, Oswald, 1919 (1), The Institute for Advanced Study, Princeton, N. J.

Vickery, Hubert Bradford, 1943 (9), Connecticut Agricultural Experiment Station, New Haven 4, Conn.

von Karman, Theodore, 1938 (4), 1501 South Marengo Avenue, Pasadena, Calif.

von Neumann, John, 1937 (1), The Institute for Advanced Study, Princeton, N. J.

Waksman, Selman Abraham, 1942 (7), Agricultural Experiment Station, New Brunswick, N. J.

Wald, George, 1950 (9), Biological Laboratories, Harvard University, 16 Divinity Avenue, Cambridge 38, Mass.

Walker, John Charles, 1945 (7), 206 Horticulture Building, University of Wisconsin, Madison 6, Wis.

Walsh, Joseph Leonard, 1936 (1), Harvard University, Cambridge 38, Mass.

Webster, David Locke, 1923 (3), Physics Department, Room 385, Stanford University, Stanford University, Calif.

Weiss, Paul Alfred, 1947 (8), Department of Zoology, University of Chicago, Chicago 37, Ill.

Went, Frits Warmolt, 1947 (7), California Institute of Technology, Pasadena 4, Calif.

Werkman, Chester Hamlin, 1946 (9), Department of Bacteriology, Iowa State College, Ames, Iowa

Wetmore, Alexander, 1945 (8), Smithsonian Institution, Washington 25, D. C.

Wever, Ernest Glen, 1940 (12), Princeton University, Princeton, N. J.

Weyl, Claus Hugo **Hermann,** 1940 (1), The Institute for Advanced Study, Princeton, N. J.

Whipple, George Hoyt, 1929 (10), School of Medicine and Dentistry, University of Rochester, 260 Crittenden Boulevard, Rochester 7, N. Y.

Whitehead, John Boswell, 1932 (4), Johns Hopkins University, Baltimore 18, Md.

Whitney, Hassler, 1945 (1), Eliot House N21, Cambridge 38, Mass.

Wigner, Eugene Paul, 1945 (3), 8 Ober Road, Princeton, N. J.

Williams, Howel, 1950 (6), Department of Geological Sciences, University of California, Berkeley 4, Calif.

Williams, Robert R., 1945 (5), 297 Summit Avenue, Summit, N. J.

Williams, Roger John, 1946 (5), Biochemical Institute, University of Texas, Austin 12, Tex.

Willier, Benjamin Harrison, 1945 (8), Department of Biology, Johns Hopkins University, Baltimore 18, Md.

Wilson, Edgar **Bright, Jr.,** 1947 (5), Harvard University, Department of Chemistry, 12 Oxford Street, Cambridge 38, Mass.

Wilson, Edwin Bidwell, 1919 (3), Harvard School of Public Health, 695 Huntington Avenue, Boston 15, Mass.

Wilson, Ralph Elmer, 1950 (2), Mt. Wilson and Palomar Observatories, 813 Santa Barbara Street, Pasadena 4, Calif.

Wilson, Robert Erastus, 1947 (4), 910 South Michigan Avenue, Chicago 80, Ill.

Wintersteiner, Oskar, 1950 (9), Squibb Institute for Medical Research, New Brunswick, N. J.

Wislocki, George Bernays, 1941 (8), Harvard Medical School, 25 Shattuck Street, Boston 15, Mass.

Wolbach, Simeon **Burt,** 1938 (10), Department of Pathology, Children's Hospital, 300 Longwood Avenue, Boston 15, Mass.

Wolfrom, Melville Lawrence, 1950 (9), Department of Chemistry, Ohio State University, Columbus 10, Ohio

Wood, Robert Williams, 1912 (3), Johns Hopkins University, Baltimore 18, Md.

Woodring, Wendell Phillips, 1946 (6), United States Geological Survey, Washington 25, D. C.

Woodworth, Robert Sessions, 1921 (12), Columbia University, New York 27, N. Y.

Wright, Frederick Eugene, 1923 (6), 2134 Wyoming Avenue, N. W., Washington 8, D. C.

Wright, Sewall Green, 1934 (8), Department of Zoology, University of Chicago, Chicago 37, Ill.

Wright, William Hammond, 1922 (2), 60 North Keeble Avenue, San Jose 11, Calif.

Wulf, Oliver Reynolds, 1949 (5), Weather Bureau Regional Office, Gates and Crellin Laboratories, California Institute of Technology, Pasadena 4, Calif.

Wyckoff, Ralph Walter Graystone, 1949 (5), Laboratory of Physical Biology, National Institutes of Health, Bethesda 14, Md.

Yerkes, Robert Mearns, 1923 (12), Yale University School of Medicine, 333 Cedar Street, New Haven, Conn.

Yost, Don Merlin Lee, 1944 (5), California Institute of Technology, Pasadena 4, Calif.

Zachariasen, Frederik William Houlder, 1949 (3), Department of Physics, University of Chicago, Chicago 37, Ill.

Zariski, Oscar, 1944 (1), Department of Mathematics, Harvard University, Cambridge 38, Mass.

Zworykin, Vladimir Kosma, 1943 (4), 103 Battle Road, Princeton, N. J.

Number of Members July 1, 1950: 461

MEMBERS EMERITI

Allen, Charles Elmer, 1924, Biology Building, University of Wisconsin, Madison 6, Wis.

Anderson, John August, 1928, P. O. Box 332, Pasadena 17, Calif.

Benedict, Francis Gano, 1914, Machiasport, Me.

Child, Charles Manning, 1935, Jordan Hall, Stanford University, Stanford University, Calif.

Dewey, John, 1910, 1158 Fifth Avenue, New York 29, N. Y.

Dickson, Leonard Eugene, 1913, Route #2, Joliet, Ill.

Fernald, Merritt Lyndon, 1935, Gray Herbarium, Harvard University, Cambridge 38, Mass.

Hektoen, Ludvig, 1918, Chicago Tumor Institute, 21 West Elm Street, Chicago 10, Ill.

Hulett, George Augustus, 1922, 44 Washington Road, Princeton, N. J.

Stratton, George Malcolm, 1928, University of California, Berkeley 4, Calif.

Whitney, Willis Rodney, 1917, General Electric Company, Schenectady 5, N. Y.

FOREIGN ASSOCIATES

The letter in parentheses following the year of election indicates the field of scientific research in which the foreign associate was working at the time of his election, as follows:

(A) Mathematics
(B) Astronomy
(C) Physics
(D) Engineering
(E) Chemistry
(F) Geology and Paleontology
(G) Botany
(H) Zoology and Anatomy
(I) Physiology and Biochemistry
(J) Pathology and Bacteriology
(K) Anthropology and Psychology

Adrian, Edgar Douglas, 1941 (I), Trinity College, Cambridge, England

Alexandroff, Paul A., 1947 (A), Mathematical Institute of the Academy of Sciences of the U.S.S.R., Bolshaya Kalushskaya 19, Moscow, U.S.S.R.

Bailey, Sir Edward, 1944 (F), 19 Greenhill Gardens, Edinburgh 10, Scotland

Bartlett, Sir Frederic Charles, 1947 (K), University of Cambridge, The Psychological Laboratory, Downing Place, Cambridge, England

Best, Charles Herbert, 1950 (I), Banting and Best Department of Medical Research, University of Toronto, Toronto, Canada

Bjerknes, V. (F. K.), 1934 (F), The University, Oslo, Norway

Bohr, Niels, 1925 (C), Institute for Theoretical Physics, Blegdamsvej 15, Copenhagen, Denmark

Bordet, Jules, 1935 (I), Pasteur Institute, Rue du Remorqueur, 28, Brussels, Belgium

Bragg, Sir William **Lawrence,** 1945 (C), Cavendish Laboratory, The University, Cambridge, England

de Broglie, Prince Louis, 1948 (C), 94 Rue Perronet, Neuilly-sur-Seine, France

Cartan, Élie, 1949 (A), University of Paris, Paris, France

Caso, Alfonso, 1943 (K), Secretaria de Bienes Nacionales e Inspeccion Administrativa, Mexico, D. F.

Chapman, Sydney, 1946 (A), Letters: Queen's College, Oxford; Printed matter: The Mathematical Institute, Oxford, England

Dale, Sir Henry Hallett, 1940 (I), The Wellcome Trust, 28 Portman Square, London, W.1, England

Debye, Peter, 1931* (C), Baker Laboratory, Cornell University, Ithaca, N. Y. (U.S.A.)

Dirac, Paul Adrien **Maurice,** 1949 (A), Department of Mathematics, St. John's College, Cambridge, England

Einstein, Albert, 1922† (C), The Institute for Advanced Study, Princeton, N. J. (U.S.A.)

Fisher, Ronald **Aylmer,** 1948 (H), Department of Genetics, University of Cambridge, 44 Storey's Way, Cambridge, England

Hadamard, Jacques, 1926 (A), 12, rue Emile Faguet, Paris XIV, France

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* Dr. Debye became a naturalized citizen in 1946 and a member of the Academy in 1947.

† Dr. Einstein became a naturalized citizen in 1941 and a member of the Academy in 1942.

Helland-Hansen, Björn, 1947 (F), Chr. Michelsens Institutt for Videnscap, Bergen, Norway

Hill, Archibald Vivian, 1941 (I), 16 Bishopswood Road, Highgate, London, N.6, England

Hill, James Peter, 1940 (H), Kanimbla, Dollis Avenue, London N.3, England

Houssay, Bernardo Alberto, 1940 (I), Viamonte 2790, Buenos Aires, Argentina

Jeffreys, Harold, 1945 (B), St. John's College, Cambridge, England

Jones, Sir Harold Spencer, 1943 (B), Royal Greenwich Observatory, Herstmonceux Castle, Hailsham, Sussex, England

Kapitza, Peter Leonidovich, 1946 (C), Institute for Physical Problems, Academy of Sciences of the U.S.S.R., Moscow, U.S.S.R.

Karrer, Paul, 1945 (E), University of Zurich, Zurich, Switzerland

Keith, Sir Arthur, 1941 (I, K), Buckston Browne Farm, Downe, Farnborough, Kent, England

Kluyver, Albert Jan, 1950 (G), Technical University, Delft, The Netherlands

Levi, Giuseppe, 1940 (H), Instituto di Anatomia Umana, Corso Massimo D'Azeglio, 52, Turin, Italy

Lim, Robert K. S., 1942 (I), Army Medical Administration, Municipal Government, Shanghai, China

Linderstrøm-Lang, Kaj Ulrik, 1947 (E), Chemical Division, Carlsberg Laboratory, Copenhagen, Denmark

Lyot, Bernard Ferdinand, 1949 (B), 9 bis rue Boileau, Paris 16, France

Pieron, Henri, 1949 (K), Institute of Psychology, University of Paris, Paris, France

Robinson, Sir Robert, 1934 (E), Dyson Perrins Laboratory, South Parks Road, Oxford, England

Ruzicka, Leopold, 1944 (E), Department of Organic Chemistry, Institute of Technology, Zurich, Switzerland

Sherrington, Sir Charles Scott, 1924 (I), Gonville and Caius College, Cambridge, England

Sommerfeld, Arnold, 1929 (C), Dunant-strasse 6, Munich 23, Germany

Southwell, Richard Vynne, 1943 (D), Imperial College of Science and Technology, South Kensington, London, S.W.7, England

Svedberg, The, 1945 (E), Fysikalisk-Kemiska Institutionen, University of Uppsala, Uppsala, Sweden

Taylor, Sir Geoffrey I., 1945 (A), Trinity College, Cambridge, England

Tiselius, Arne W. K., 1949 (I), Institute of Biochemistry, Uppsala University, Uppsala, Sweden

Vallee-Poussin, C. de la, 1929 (A), University of Louvain, Louvain, Belgium

Vening Meinesz, Felix Andries, 1939 (F), Potgieterlaan 5, Amersfoort, The Netherlands

Watson, D. M. S., 1938 (H), University College, Gower Street, London, W.C.1, England

Wieland, Heinrich, 1932 (E), Sophienstrasse 9, Munich 2 NW, Germany
Winge, Øjvind, 1949 (I), Department of Physiology, Carlsberg Laboratory, Copenhagen (Valby), Denmark
Yukawa, Hideki, 1949 (C), Department of Physics, Columbia University, New York 27, N. Y. (U.S.A.)

Number of Foreign Associates July 1, 1950: 46.

If a foreign associate becomes a member of the Academy his name is not counted in the limit of 50 foreign associates.

SECTIONS

(1) *Mathematics*—28 members

Zariski, Oscar, *Chairman* (1952)	Evans, G. C.	Smith, Paul A.
Albert, A. A.	Kasner, Edward	Stone, M. H.
Alexander, J. W.	Lefschetz, Solomon	Thomas, T. Y.
Bell, E. T.	McShane, E. J.	Vandiver, H. S.
Bliss, G. A.	Mac Lane, Saunders	Veblen, Oswald
Bochner, S.	Miller, G. A.	von Neumann, John
Coble, A. B.	Moore, R. L.	Walsh, J. L.
Douglas, Jesse	Morse, Marston	Weyl, Hermann
Eisenhart, L. P.	Murnaghan, F. D.	Whitney, Hassler
	Ritt, J. F.	

(2) *Astronomy*—28 members

Merrill, P. W., *Chairman* (1953)	King, A. S.	Russell, H. N.
Abbot, C. G.	Kuiper, G. P.	Seares, F. H.
Adams, W. S.	Lambert, W. D.	Shapley, Harlow
Aitken, R. G.	Leuschner, A. O.	Slipher, V. M.
Babcock, H. D.	Mayall, N. U.	Stebbins, Joel
Bowen, I. S.	Menzel, D. H.	Struve, Otto
Fleming, J. A.	Mitchell, S. A.	Trumpler, R. J.
Hubble, E. P.	Moulton, F. R.	Wilson, Ralph E.
Joy, A. H.	Nicholson, S. B.	Wright, W. H.
	Ross, F. E.	

(3) *Physics*—65 members

Beams, J. W., *Chairman* (1951)	Bloch, Felix	Davisson, C. J.
Allison, S. K.	Breit, Gregory	DuBridge, L. A.
Alvarez, L. W.	Bridgman, P. W.	Dunning, J. R.
Anderson, C. D.	Brode, R. B.	Einstein, Albert
Bacher, R. F.	Coblentz, W. W.	Epstein, P. S.
Bainbridge, K. T.	Compton, A. H.	Fermi, Enrico
Berkner, L. V.	Compton, K. T.	Franck, James
Bethe, H. A.	Condon, E. U.	Goudsmit, S. A.
Birge, R. T.	Coolidge, W. D.	Houston, W. V.
Bjerknes, Jacob	Crew, Henry	Hull, A. W.
	Davis, Bergen	Ives, H. E.

Kemble, E. C.
Lauritsen, C. C.
Lawrence, E. O.
Loomis, F. W.
Lyman, Theodore
McMillan, E. M.
Mason, Max
Millikan, R. A.
Mulliken, R. S.
Nier, A. O. C.
Oppenheimer, J. R.
Pegram, G. B.
Pierce, G. W.
Piggot, C. S.
Rabi, I. I.
Reichelderfer, F. W.
Rossby, C.-G.
Rossi, Bruno
Saunders, F. A.
Schwinger, Julian
Slater, J. C.
Slichter, L. B.
Stern, Otto
Stewart, G. W.
Sverdrup, H. U.
Teller, Edward
Tuve, M. A.
Van Vleck, J. H.
Webster, D. L.
Wigner, E. P.
Wilson, Edwin B.
Wood, R. W.
Zachariasen, W. H.

(4) *Engineering*—36 members

Hunsaker, J. C., *Chairman* (1953)
Adams, C. A.
Briggs, L. J.
Buckley, O. E.
Bush, Vannevar
Cochrane, E. L.
Curme, G. O., Jr.
Dryden, H. L.
Dunn, Gano
Durand, W. F.
Fletcher, Harvey
Foote, P. D.
Gibbs, W. F.
Gilliland, E. R.
Herty, C. H., Jr.
Hoover, Herbert
Jeffries, Zay
Kelly, M. J.
Kettering, C. F.
Lewis, W. K.
Loomis, A. L.
Merica, P. D.
Murphree, E. V.
Raymond, A. E.
Savage, J. L.
Slepian, Joseph
Soderberg, C. R.
Stratton, J. A.
Suits, C. G.
Terman, F. E.
Thomas, C. A.
Timoshenko, Stephen
von Karman, T.
Whitehead, J. B.
Wilson, Robert E.
Zworykin, V. K.

(5) *Chemistry*—62 members

Fuson, R. C., *Chairman* (1953)
Adams, L. H.
Adams, Roger
Bachmann, W. E.
Bancroft, W. D.
Bartlett, P. D.
Baxter, G. P.
Bogert, M. T.
Bolton, E. K.
Conant, J. B.
Cope, A. C.
Craig, L. C.
Daniels, Farrington
Debye, Peter
Elderfield, R. C.
Eyring, Henry
Fieser, L. F.
Folkers, Karl
Giauque, W. F.
Gilman, Henry
Hammett, L. P.
Harkins, W. D.
Harned, H. S.
Hildebrand, J. H.
Hudson, C. S.
Ipatieff, V. N.
Jacobs, W. A.
Johnson, J. R.
Keyes, F. G.
Kharasch, M. S.
Kirkwood, J. G.
Kistiakowsky, G. B.
Kraus, C. A.
La Mer, V. K.
Lamb, A. B.
Langmuir, Irving
Latimer, W. M.
Libby, W. F.
Lind, S. C.
Longsworth, L. G.
McElvain, S. M.
MacInnes, D. A.
Marvel, C. S.
Mayer, J. E.
Mees, C. E. K.
Noyes, W. A., Jr.
Onsager, Lars
Pauling, Linus
Pitzer, K. S.
Rodebush, W. H.
Scatchard, George
Schlesinger, H. I.
Seaborg, G. T.
Small, L. F.
Smith, L. I.
Urey, H. C.
Williams, Robert R.
Williams, Roger J.
Wilson, E. Bright, Jr.
Wulf, O. R.
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